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Search Results -

Terms	Documents
L10 and @RLAD<19991104	47

Database: US Patents Full-Text Database; US Pre-Grant Publication Full-Text Database; JPO Abstracts Database; EPO Abstracts Database; Derwent World Patents Index; IBM Technical Disclosure Bulletins

Search: L11

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Search History

DATE: Thursday, December 19, 2002 Printable Copy Create Case

Set Name side by side	Query	Hit Count	Set Name result set
DB=USPT,PGPB,DWPI; THES=ASSIGNEE; PLUR=YES; OP=OR			
L11	L10 and @RLAD<19991104	47	L11
L10	L9 and peptide and arthritis	95	L10
L9	Hsp60 or ((60 or 60kD or (60 adj1 kilodalton)) near5 (heat adj1 shock))	385	L9
L8	L7 and @RLAD<19991104	64	L8
L7	L6 and peptide	95	L7
L6	L4 and arthritis	105	L6
L5	L4 and peptide	189	L5
L4	Hsp65 or ((65 or 65kD or (65 adj1 kilodalton)) near5 (heat adj1 shock))	228	L4
L3	Hsp65 or (65?? near5 (heat adj1 shock))	135	L3
L2	Hsp65 or (65 near5 (heat adj1 shock))	212	L2
L1	Hsp65	114	L1

END OF SEARCH HISTORY

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     (FILE 'HOME' ENTERED AT 11:03:30 ON 19 DEC 2002)

     FILE 'REGISTRY' ENTERED AT 11:03:36 ON 19 DEC 2002
L1             0 S GPKGRNVVLEKKWGAPTITNDG

     FILE 'MEDLINE, CAPLUS, EMBASE, BIOSIS' ENTERED AT 11:04:39 ON 19 DEC 2002
L2           234 S NAPARSTEK Y/AU
L3           114 DUP REM L2 (120 DUPLICATES REMOVED)
L4            14 S L3 AND ARTHRITIS

     FILE 'REGISTRY' ENTERED AT 11:06:42 ON 19 DEC 2002
L5             0 S *GPKGRNVVLEKKWGAPTITNDG*

     FILE 'MEDLINE, CAPLUS, EMBASE, BIOSIS' ENTERED AT 11:08:06 ON 19 DEC 2002
L6         84997 S HSP60 OR HSP65 OR (HEAT (1W) SHOCK)
L7          3673 S L6 AND MYCOBACTER?
L8           716 S L7 AND ARTHRITIS
L9           307 DUP REM L8 (409 DUPLICATES REMOVED)
L10          799 S L7 AND PEPTIDE
L11          140 S L10 AND ARTHRITIS
L12           61 DUP REM L11 (79 DUPLICATES REMOVED)
L13           46 S L12 AND PY<1999

     FILE 'REGISTRY' ENTERED AT 11:18:18 ON 19 DEC 2002
L14            1 S THR-PHE-GLY-LEU-GLN-LEU-GLU-LEU-THR
L15            0 S
GLY-PRO-LYS-GLY-ARG-ASN-VAL-VAL-LEU-GLU-LYS-LYS-TRP-GLY-ALA-P
L16            0 S
(GLY-PRO-LYS-GLY-ARG-ASN-VAL-VAL-LEU-GLU-LYS-LYS-TRP-GLY-ALA-
L17            0 S
(GLY-PRO-LYS-GLY-ARG-ASN-VAL-VAL-LEU-GLU-LYS-LYS-TRP-GLY-ALA-

     FILE 'MEDLINE, CAPLUS, EMBASE, BIOSIS' ENTERED AT 11:24:12 ON 19 DEC 2002
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L13 ANSWER 34 OF 46 MEDLINE
ACCESSION NUMBER: 90171847 MEDLINE
DOCUMENT NUMBER: 90171847 PubMed ID: 1689764
TITLE: Recognition of a **mycobacteria**-specific epitope in the 65-kD **heat-shock** protein by synovial fluid-derived T cell clones.
AUTHOR: Gaston J S; Life P F; Jenner P J; Colston M J; Bacon P A
CORPORATE SOURCE: Department of Rheumatology, University of Birmingham, United Kingdom.
SOURCE: JOURNAL OF EXPERIMENTAL MEDICINE, **(1990 Mar 1)** 171 (3) 831-41.
Journal code: 2985109R. ISSN: 0022-1007.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199004
ENTRY DATE: Entered STN: 19900601
Last Updated on STN: 19960129
Entered Medline: 19900412

AB Adjuvant **arthritis** in rats is induced by a T cell clone specific for amino acids 180-188 of the **mycobacterial** 65-kD **heat -shock** protein, and synovial T cell responses to this same Ag have been noted in human **arthritis**. We have isolated 65-kD Ag-specific T cell clones from synovial fluid mononuclear cells of a patient with acute **arthritis**, which, unlike the corresponding PBMC, showed a marked proliferative response to the 65-kD Ag. Using synthetic **peptides** corresponding to the whole sequence of the 65-kD Ag, all the clones were shown to recognize an epitope present in the first NH2-terminal **peptide** (amino acids 1-15), with no response to the adjacent **peptide** (amino acids 6-22) or to any other **peptide**. The complete dominance of this epitope in the response to the 65-kD Ag was shown by documenting responses to the **peptide** in PBMC obtained after recovery from the **arthritis**. This epitope, like that recognized by the rat arthritogenic T cell clone, is in a portion of the 65-kD sequence that is not conserved between bacteria and eukaryotes, so that in this case, joint inflammation could not be attributed to bacteria-induced T cell clones cross-reacting with the self 65-kD Ag.

L13 ANSWER 15 OF 46 MEDLINE
ACCESSION NUMBER: 94289341 MEDLINE
DOCUMENT NUMBER: 94289341 PubMed ID: 7517177
TITLE: Differential rat T cell recognition of cathepsin D-released fragments of **mycobacterial** 65 kDa **heat-shock** protein after immunization with either the recombinant protein or whole **mycobacteria**.
AUTHOR: van Noort J M; Anderton S M; Wagenaar J P; Wauben M H; van Holten C; Boog C J
CORPORATE SOURCE: Medical Biological Laboratory TNO, Rijswijk, The Netherlands.
SOURCE: INTERNATIONAL IMMUNOLOGY, **(1994 Apr)** 6 (4) 603-9. Journal code: 8916182. ISSN: 0953-8178.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199408
ENTRY DATE: Entered STN: 19940815
Last Updated on STN: 19960129
Entered Medline: 19940804

AB T cells specific for the **mycobacterial** 65 kDa **heat-shock** protein (**hsp65**) play a pivotal role in the development of adjuvant **arthritis** (AA) in Lewis rats. Upon adoptive transfer, CD4+ T cells recognizing a particular **hsp65** epitope trigger the onset of disease. Activation of **hsp65**-reactive T cells can be achieved by immunization with heat-killed **mycobacteria** in mineral oil--complete Freund's adjuvant (CFA)--or with purified recombinant **hsp65**. **Arthritis**, however, will only develop after immunization with CFA. In fact, preimmunization with **hsp65** protects against any subsequent attempt to induce AA. In this study, we examined polyclonal lymph node cell responses in Lewis rats, immunized with either CFA or purified recombinant **hsp65** in incomplete Freund's adjuvant, to a set of **hsp65** fragments generated by a mild digestion with cathepsin D. Proliferative responses to several **hsp65** fragments varied with the type of antigen used for immunization. A cathepsin D-released fragment, identified as residues 376-408, preferentially triggered proliferation of rat T cells after **hsp65** immunization. Preimmunization of Lewis rats with this **peptide** delayed the onset and reduced the severity of AA. Preimmunization with another fragment which was preferentially recognized after CFA immunization, representing residues 40-60, did not have such a protective effect. Our findings suggest the presence of **mycobacterial hsp65** determinants that selectively trigger AA-regulating T cells and illustrate that cathepsin D may be used as an experimental tool to generate such determinants.

L13 ANSWER 7 OF 46 MEDLINE
ACCESSION NUMBER: 97226230 MEDLINE
DOCUMENT NUMBER: 97226230 PubMed ID: 9082939
TITLE: Clonal expansion of **mycobacterial heat-shock** protein-reactive T lymphocytes in the synovial fluid and blood of rheumatoid **arthritis** patients.
AUTHOR: Celis L; Vandevyver C; Geusens P; Dequeker J; Raus J; Zhang J
CORPORATE SOURCE: Willems-Instituut, Diepenbeek, Belgium.
SOURCE: ARTHRITIS AND RHEUMATISM, **(1997 Mar)** 40 (3) 510-9.
Journal code: 0370605. ISSN: 0004-3591.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199704
ENTRY DATE: Entered STN: 19970414
Last Updated on STN: 19970414
Entered Medline: 19970403

AB OBJECTIVE: To examine the reactivity pattern and T cell receptor (TCR) characteristics of **mycobacterial heat-shock** protein 65 (**hsp65**)-reactive T cells generated from paired synovial fluid (SF) and peripheral blood (PB) samples obtained from rheumatoid **arthritis** (RA) patients and from healthy subjects. METHODS: The reactivity pattern of **hsp65**-reactive T cell clones generated under limiting-dilution conditions was analyzed in 3H-thymidine incorporation assays. The TCR variable regions of these **hsp65**-reactive T cells were characterized by polymerase chain reaction with TCR AV- and BV-specific primers and by DNA sequence analysis of the third complementarity-determining region (CDR3). RESULTS: The **hsp65**-reactive T cells derived both from RA patients and controls preferentially recognized the 1-170 and 303-540 regions of **hsp65** and did not cross-react with human **hsp60**. The **hsp65**-reactive T cell clones derived from RA patients displayed a restricted TCR AV and BV gene usage, which can be attributed to the limited clonal origin(s) of the independent T cell clones, as evidenced by CDR3 sequence analysis. These clonally expanded T cells were found in both PB and SF and in different inflamed joints of RA patients. CONCLUSION: Our study suggests that there is in vivo clonal activation and expansion of **mycobacterial hsp65**-reactive T cells in patients with RA.